TABLE 4.—Intervention, followup, and cessation results for three major community prevention trials

Community trial	Intervention	Control group contact	Followup	Reported cessation rates/ (objective measures)		
				Treated	Control	Time
WHO European Collaborative Trial: United Kingdom (62)	Mass media intervention for all factory workers Antismoking clinics for all smokers High risk smokers (top 10–15% risk) offered individual treatment (four 15 min sessions in year 1 with company physician)	Random 10% sample invited for screening The rest of control males not told of their participation in the trial	Random 5% IG examined yearly All survivors examined at end of trial Same random 10% CG screened, reexamined at 2 years	12%[1] (high risk smokers) 9% (all smokers) 7% (non-high-risk smokers) (no objective measures used)	(no change)	5 yrs
WHO European Collaborative Trial: Belgium (62)	Mass media intervention for all factory workers High risk smokers (top 21% risk) offered counseling and examination by project physicians twice per year	Random 10% invited for screening Other 90% had resting ECG only	Random 5% IG examined yearly All survivors examined at end of trial	18.7%[2] (high risk smokers) 12.5% (all smokers) (no objective measures used)	12.2% (high risk smokers) 12.6% (all smokers)	2 yrs

TABLE 4.—Continued.

Community trial	Intervention	Control group contact	Followup	Reported cessation rates/ (objective measures)		
North Karelia (Finland) Project (*52, 53, 63*)	Comprehensive "community action" program against risk factors All forms of media used Special groups set up as needed	6.6% random sample examined at baseline and 5 years	Random 6.6% of each community surveyed and examined in 1977 Results compared to assess RF change	17%[3] (male smokers)	15% (male smokers) (SCN drawn for random subsamples) (Correlations reported, but adjustments not made)	5 yrs

[1] $P \leq 0.01$.
[2] $P \leq 0.05$.
[3] Not significant.

intervention subjects in the top 21 percent of the risk score distribution were placed in the high risk group (n=1,601).

All smokers in the intervention factories received the mass media approach previously noted for the WHO trials, and all family and factory physicians received regular information about the participants' risk factors and took part in intervention (*8, 26, 27*). Twice a year, high risk subjects were individually counseled and examined by two project physicians (*8, 26, 27*) (Table 3).

Reports of the smoking results for this trial have included comparisons of the results for the 5 percent random sample in the intervention group with the results for the 10 percent random sample of the control group at 2-year followup and comparisons of the results of the high risk subjects in the intervention group with the results of the high risk subjects selected from the 10 percent random sample of the control group screened at baseline (*9, 26, 27*).

Reported smoking rates have not been validated with objective measures. Among the high risk smokers, 18.7 percent in the intervention group and 12.2 percent in the control group reported cessation at 2 years, producing a statistically significant difference ($p \leq 0.05$). For the random samples there was no difference in reported cessation, with approximately 12.5 percent of the smokers reporting cessation in both groups (*26, 27*). Smoking cessation rates for the intervention group at 1 year was 12 percent and 8 percent for the high risk subjects and the random sample, respectively (*27*), indicating that cessation occurred gradually over the 2-year period.

The North Karelia (Finland) Project

The North Karelia project was carried out in Finland during 1972–1977 as a comprehensive community program to study the control of cardiovascular disease (CVD), with special emphasis on CHD, by reduction of the major alterable CHD risk factors (smoking, increased serum cholesterol, and hypertension (*52, 53, 54, 63, 64, 65*). The intervention area was the county of North Karelia in eastern Finland, which had the highest rates of CVD in that country. The county of Kuopio, also in eastern Finland, was selected as the control area because of its similarity to North Karelia.

Both in 1972 and in 1977 a representative random 6.6 percent sample of the population born between 1913 and 1947 (aged 25 to 59 in 1972 and 30 to 74 in 1977) was drawn from the two counties by using the national population register (*53, 54*). The samples in 1972 and in 1977 were independent of each other. Those persons surveyed were sent a letter explaining the study, a questionnaire assessing medical history, health behavior and attitudes, attempts to change health behavior, and stress and an invitation and date for a physical examination. Over 10,000 subjects were studied each time, with a participation rate of about 90 percent (*52, 53, 54, 63, 64, 65*).

The comprehensive "community action" program against risk factors was integrated into the health and social services of the county and was aimed at primary and secondary prevention, although primary prevention was emphasized. Public information was provided through newspapers, radio, leaflets, posters, health education meeetings, and campaigns at schools and places of work (*53*); new services were set up if needed, personnel were trained, and environmental changes (e.g., smoking restrictions) were implemented. The project team planned the activities, prepared the educational material, helped train personnel, and got the community into action (*54*). Smoking cessation group activities were available to those smokers who wanted them, on the basis of a 3-week model developed by the project (*63*). Approximately 55 percent of the smokers were willing to join the groups, and 71 percent of those who started completed the groups (*63*). Approximately 27 percent of those who started the group reported smoking cessation at 6 months.

The outcomes concerning changes in smoking are based on the comparison of data obtained in the baseline survey and in the 5-year terminal survey from the study community and the matched control community. The validity of the self-reports of smoking behavior was tested on a random subsample of subjects who were given a second interview about smoking by trained nurses unaware of the answers to the survey questionnaire (*53*). When classified by an interval of 5 or 10 cigarettes, the agreement between the two results was 93 and 97 percent, respectively. The agreement was 99 percent when classification was smoker or nonsmoker (*53*). Serum thiocyanate (SCN) determinations made during the termination interview provided further validation. Since it is not otherwise noted in the scientific reports, it is assumed that the results are based on self-report and are not corrected. Individuals who reported ever having smoked regularly or having smoked during the preceding month on an average of more than once a day were classified as smokers. The reported number of cigarettes, cigars, and pipes smoked per day was calculated as the amount smoked (*53*).

The prevalence of smoking in the study and in the control area for men was 50.2 and 59.9 percent, respectively, and for women 11.7 and 13.1 percent at the start of the study. At year 5, 17 percent of the baseline male smokers in North Karelia reported smoking cessation and 15 percent of the baseline male smokers in Kuopio reported cessation of smoking (*52, 53*) (Table 4). Thus, smoking had decreased considerably in both the control and the study groups, yielding a nonsignificant net reduction in North Karelia of 2.5 percent for the men and 6.1 percent for the women. With regard to amount smoked, North Karelian men smoked more than did the men in the control area in 1972 (9.9 versus 8.9 cigarettes per day), and both groups were smoking 8.1 cigarettes per day by the end of the study (*53*), producing

a significant net reduction among North Karelian men of 9.8 percent. The mean number of cigarettes smoked by smokers in North Karelia was 19 cigarettes per day, which remained stable during the study (*63*).

A small net reduction occurred in the prevalence of smoking because, even though considerable cessation was reported in North Karelia, smoking also decreased at a similar rate in Kuopio, the control area. The investigators noted several possible explanations for this decrease (*53*). There was an increase in interest in antismoking activities toward the end of the study period: the Finnish Parliament passed new antismoking legislation in 1976, and a new medical school opened in Kuopio in 1972. They also indicated on the basis of internal followup surveys that most of the reduction in smoking occurred at the beginning of intervention in North Karelia, after the first intensive public antismoking campaign, and that this lower level of smoking was maintained during the rest of the period (*39, 63*).

Deficiencies in the Community Preventive Trials

The major deficiencies in these community preventive trials are the same as those noted for the clinical trials; i.e., lack of objective data to verify self-reported outcomes, use of cross-sectional analyses to the almost complete exclusion of cohort analyses, failure to provide sufficient information in scientific reports to allow adequate interpretation of outcomes, and lack of evaluation of components of the intervention packages.

Objective data to verify self-reports were not used in the United Kingdom and the Belgium heart disease prevention projects, and although SCN was measured in the North Karelia study, it was not used to adjust the self-reported cessation data. Data for strata of smokers by age were presented for the North Karelia study (*74*), but not for cohorts of smokers by smoking-behavior-change categories. Longitudinal data for cohorts of smokers in the other two trials were not presented. The value of cohort data is illustrated by a statement made by the North Karelia investigators in which they noted that even though the smoking cessation rates were similar in North Karelia and Kuopio, most of the cessation in North Karelia occurred at the beginning of the project and was maintained (*63, 74*). This information was obtained by the use of followup surveys of samples of residents. It was hypothesized that most of the cessation for the comparison community may have occurred near the termination of the project when antismoking legislation and other changes had occurred there (*39, 63*). Cohort data for the comparison community or for subgroups are not available; thus, the hypothesis cannot be tested.

Use of the same subjects for baseline and termination surveys are likely to influence outcomes; therefore, the change may look better than it actually is (*53, 54, 63*). The use of a cohort design might therefore produce a net effect that is not totally a consequence of the intervention alone, but may also include the effect of the first survey as well as its interaction with the intervention (*63*). Thus, there is a possible need to examine independent cross-sectional population samples in the two areas under study at the start and termination of the project. This hypothesis is not supported by data from the North Karelia study, where cessation rates for 6.6 percent of the random sample of smokers in the baseline survey of the control community who were also included in the termination survey were similar to the rates for the rest of the smokers who were surveyed only at termination (*63*). Data from the United Kingdom heart disease prevention project (*62*) also failed to support the hypothesis of a possible intervention effect from screening.

Different components of intervention were not differentially evaluated within any of the community trials because of the community orientation of the projects. Thus, conclusions about the relative contributions of different programs, subprograms, or channels of action cannot be drawn (*39*).

Comparison of Community Trials Outcomes

As is noted in Table 4, the 2-year cessation data for both the Belgium and the United Kingdom WHO trials demonstrate that there were significant differences in reported cessation rates for the intensively intervened-with high risk smokers as compared with the smokers in the control factories, but there were no significant differences between the non-high-risk smokers in the intervention factories who received a media-only approach as compared with the non-high-risk smokers in the control factories. This outcome is similar to the previously noted finding in the Stanford study; i.e., media only had no more intervention impact than had no intervention. This lack of demonstrated impact for a media approach to smoking cessation in the Belgium study was in part due to the 12.5 percent cessation rate achieved by the control group. This occurrence of cessation in a control group is similar to that demonstrated in each of the clinical trials. Again, a saturation point may have been reached in groups in which there is already an increased level of awareness, and intensive intervention may be necessary if additional cessation is to be realized in the next level of smokers. It may also be, as previously noted, that although cessation occurred among the nonintervention smokers, the long-term maintenance rate among those in this group who stop smoking may be significantly different from the long-term maintenance rate for the intervention group

smokers. Because of the lack of cohort data, this issue cannot be addressed.

The range of cessation rates among the comparison groups for the three trials is large, 0 to 15 percent, with the lowest rate recorded for the United Kingdom group of the WHO collaborative study and the highest for the Belgium group of the same study. The 0 percent cessation rate for the control group in the United Kingdom trial is puzzling, as this is less than the spontaneous cessation rate observed in the general population. More in-depth analysis of the data for the 10 percent random sample of the control group who were screened at baseline and reexamined at 2 years is indicated. Different protocols for contact with the control group were used for the United Kingdom from those used for the Belgium groups. The most notable difference was that 90 percent of the United Kingdom control group were not told of their participation in the trial, but 90 percent of the Belgium group were told of their participation and had a resting ECG. An additional 10 percent were told of their participation and had complete physical examinations. It can be hypothesized that the use of an ECG may have had an intervention effect for the Belgium control group. The 15 percent rate for the North Karelia study was determined at 5 years. One might hypothesize that this rate would be lower at 2 years, the point at which the other two studies conducted followup.

The 5-year cessation rate for the United Kingdom collaborative trial is also low when compared with the 2-year rate for the Belgium collaborative trial, which utilized a similar protocol. A major difference for these two groups was that high risk smokers in the Belgium study received two examinations per year while those in the United Kingdom were given one. There was also a difference in the number of physician-intervention visits during year 1; two were used in the Belgium study and four were used in the United Kingdom trial. Perhaps the feedback provided by an examination has a greater intervention effect than a session with a physician intended for counseling only. It can also be hypothesized that cultural differences may have affected the differences in outcome between the two groups in the same trial.

In general, the use of community programs that used only a media approach did not produce a greater intervention effect than was observed in the comparison community. The incorporation of more intensive intervention in groups in addition to the media approach was necessary before significant differences could be realized. The same outcomes were observed in the Stanford study.

Conclusions

1. Smokers involved in intervention programs demonstrate higher smoking cessation rates than those in control groups.
2. In general, the success of smoking intervention programs is related to the amount of intervention provided.

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SECTION 8. THE EFFECT OF CIGARETTE SMOKING CESSATION ON CORONARY HEART DISEASE

Epidemiologic Evidence Regarding Smoking Cessation and Coronary Heart Disease

The epidemiologic data on smoking and coronary heart disease (CHD) were reviewed in detail in a preceding section, as well as in the Reports of the Surgeon General for 1964, 1971, and 1979 (*60, 61, 62*). Coronary heart disease (ICD/6 and ICD/7 No. 420) before 1968 and ischemic heart disease (ICD/8 and ICD/9 Nos. 410–414) since 1968 are considered synonymous with one another for all practical purposes and are abbreviated as CHD. Terminology and data on CHD are discussed in detail elsewhere (*33, 34, 44*). This discussion is limited to epidemiologic data on smoking cessation and CHD. Several prospective studies involving self-selected questionnaire respondents include extensive epidemiologic data on smoking cessation and CHD mortality. The results, summarized in Table 1, show CHD death rates for former smokers relative to never smokers as a function of the number of years stopped smoking cigarettes, generally determined as of the time the questionnaire was completed. Data in this form are available only for men, generally white men. The studies are as follows: the British physicians study, including 10-year followup (*10, 11*) and 20-year followup (*12*); the American Cancer Society 9-State study (*22, 23*) and the American Cancer Society 25-State cancer prevention study (*20, 21*); the U.S. veterans study with 8.5-year followup (*32*) and 16-year followup (*49*); and the Swedish representative sample study with 10-year followup (*4*). Excluded were numerous studies that present data only on former smokers as a whole or have data on a few special categories of former smokers, such as Shapiro et al. (*58*) and Hirayama and Hamano (*25*). Much of these other epidemiologic data on former smokers is summarized in the 1979 Report of the Surgeon General on Smoking and Health (*60*) and in the preceding section of this Report on coronary heart disease.

Numerous epidemiologic studies (*10, 11, 12, 14, 20, 21, 32*) have shown a decrease in CHD mortality for ex-smokers compared with continuing smokers, and it has been suggested that smoking cessation accounts for this salutary effect. Another view (*57*) has been that continuing smokers and quitters are somehow constitutionally different and that their health experiences might also be different, independent of smoking status. Two prospective studies of current smokers, some of whom became persistent quitters during the course of the study, show that persistent quitters have lower CHD and total death rates than do continuing smokers (*17, 18*). Friedman et al. (*17*) examined this question in some detail in the Kaiser-Permanente study of over 25,000 persons. They compared 18 baseline characteristics related to coronary disease in quitters and continuing smokers at a time when all were smoking. They found that the beneficial effects of quitting on CHD mortality could not be explained by differences in their baseline characteristics (Table 2).

TABLE 1.—Male coronary heart disease and total mortality ratios for current and former cigarette smokers relative to never smokers, as a function of years stopped smoking

Overall cohort description	Smoking selection criteria	Years stopped[1]	Coronary heart disease mortality ratio[2]			All causes mortality ratio[2]		
British physicians[3]	Cigarettes only	0	1.41 (464)			1.37 (1566)		
		1–4	1.05 (28)			0.96 (71)		
		5–9	1.25 (61)			1.18 (204)		
		10–14	1.16 (59)			1.12 (204)		
		15+	1.12 (40)			1.11 (153)		
		NS	1.00 (113)			1.00 (436)		
			Attained age			Attained age		
			30–54	55–64	65+	30–64	65+	30+
British physicians[4]	Cigarettes only, at least 5 years	0	3.5	1.7	1.3	2.0	1.6	1.8
		1–4	1.9 (7)	1.9 (19)	1.0 (24)	1.7 (67)	1.4 (99)	1.5 (166)
		5–9	1.3 (10)	1.4 (34)	1.3 (76)	1.6 (141)	1.4 (242)	1.5 (383)
		10–14	1.4 (10)	1.7 (38)	1.2 (62)	1.4 (104)	1.2 (206)	1.3 (310)
		15+	1.3 (7)	1.3 (45)	1.1 (148)	1.1 (106)	1.1 (484)	1.1 (590)
		NS	1.0 (32)	1.0 (75)	1.0 (182)	1.0 (326)	1.0 (611)	1.0 (937)
			Cigarettes smoked per day			Cigarettes smoked per day		
			1–19	20+		1–19	20+	
American Cancer Society 9-State study[5]	Cigarettes only	0	1.75 (604)	2.20 (604)		1.61 (2303)	2.02 (1326)	
		<1	2.10 (23)	3.00 (18)		2.04 (51)	2.69 (35)	
		1–9	1.54 (80)	2.06 (64)		1.30 (159)	1.82 (135)	
		10+	1.09 (40)	1.60 (40)		1.08 (141)	1.50 (87)	
		NS	1.00 (709)	1.00 (709)		1.00 (1644)	1.00 (1644)	

TABLE 1.—Continued.

Overall cohort description	Smoking selection criteria	Years stopped[1]	Coronary heart disease mortality ratio[2]		All causes mortality ratio[2]	
			Cigarettes smoked per day for initial ages 40–79		Cigarettes smoked per day for initial ages 50–74	
			1–19	20+	1–19	20+
American Cancer Society 25-State study[6]	Cigarettes only	0	1.90 (1063)	2.55 (2822)	1.72 (2015)	1.94 (3741)
		<1	1.62 (29)	1.61 (62)	1.61 (64)	2.18 (213)
		1–4	1.22 (57)	1.51 (154)	1.44 (144)	1.98 (499)
		5–9	1.26 (55)	1.16 (135)	1.34 (128)	1.49 (416)
		10–19	0.96 (52)	1.25 (133)	1.02 (255)	1.32 (546)
		20+	1.08 (70)	1.05 (80)		
		NS	1.00 (1841)	1.00 (1841)	1.00 (3512)	1.00 (3512)
			Attained age, 55–64		Attained age, 55–64	
U.S. veterans[7]	Cigarettes with or without cigars/pipes, stopped for other than doctor's orders	0	1.66 (3064)		1.72 (6928)	
		1–4	1.34 (155)		1.56 (379)	
		5–9	1.47 (279)		1.42 (596)	
		10–14	1.13 (161)		1.28 (365)	
		15+	0.97 (342)		1.07 (779)	
		NS	1.00 (1218)		1.00 (2617)	
			Attained age, 31–99		Attained age, 31–99	
U.S. veterans[8]	Cigarettes with or without cigars/pipes, stopped for other than doctor's orders	0	1.58 (13,845)		1.73 (36,143)	
		1–4	1.35 (150)		~1.5 (384)	
		5–9	1.38 (599)		~1.4 (1441)	
		10–14	1.29 (997)		~1.3 (2445)	
		15–19	1.21 (1101)		~1.2 (2767)	
		20+	1.05 (2418)		~1.05 (6049)	
		NS	1.00 (~6500)		1.00 (16,224)	

TABLE 1.—Continued.

Overall cohort description	Smoking selection criteria	Years stopped [1]	Coronary heart disease mortality ratio [2]			All causes mortality ratio [2]		
			Years smoked cigarettes, initial age, 40–69			Years smoked cigarettes, initial age, 40–69		
			< 20	≥ 20	Total	< 20	≥ 20	Total
Swedish representative sample [9]		0			1.7 (212)			1.4 (557)
		1–9	0.9 (7)	1.6 (84)	1.5 (97)	1.0 (26)	1.4 (212)	1.3 (253)
		10+	0.9 (40)	1.1 (46)	1.0 (86)	1.0 (123)	1.0 (117)	1.0 (241)
		NS	1.0 (219)	1.0 (219)	1.0 (219)	1.0 (671)	1.0 (671)	1.0 (671)

[1] Years stopped smoking was measured as of beginning of followup, except for the U.S. veterans study, where the number of years stopped was increased by 1 with the passage of each calendar year unless death occurred. 0 years stopped denotes current smoker; NS denotes never smoker.

[2] Mortality ratio is former smoker death rate relative to never smoker death rate, properly adjusted for age; ratio for never smokers is defined to be 1.0. Number of deaths are in parentheses.

[3] Study of 34,445 men aged 20+, at 10-year followup, 1951–1961. Doll and Hill (*10, 11*).

[4] Study of 34,440 men aged 20+, at 20-year followup, 1951–1971. Doll and Peto (*12*).

[5] Study of 187,783 men aged 50–69, at 44-month followup, 1952–1955. Hammond and Horn (*22, 23*).

[6] Study of 440,558 men aged 30+, approximately at 4-year followup, 1959–1963, for total mortality, and 358,534 disease-free men at 6-year followup, 1959–1965, for CHD mortality Hammond (*20*), Hammond and Garfinkel (*21*).

[7] Study of 248,046 men aged 31–84, at 5.5-year or 8.5-year followup, 1954–1962. Kahn (*32*).

[8] Study of 248,045 men aged 31–84, at 13-year or 16-year followup, 1954–1969. Rogot and Murray (*49*).

[9] Study of 51,911 men aged 18–69, at 10-year followup, 1963–1972. Cederlof et al. (*4*).

People who persisted in cigarette smoking had more than twice the risk of dying from CHD than those who quit even after taking into account the other baseline differences. These studies provide stronger evidence regarding the benefits of quitting than do the studies in which all of the ex-smokers had stopped smoking before the beginning of the followup.

Data from two "natural experiments" of smoking cessation among physicians in Britain (*12*) and in California (*14*) are presented in Table 3. Because these physicians have stopped smoking to a much greater extent than has the general male population, the subsequent CHD mortality trend in physicians as a whole relative to the general population constitutes a crude estimate of the overall mortality benefits of smoking cessation. This assumes that there have been no other major risk factor changes in the compared populations, but unfortunately, other risk factors were not measured in these two studies. Both studies support the earlier prospective studies with regard to the benefits of smoking cessation on CHD mortality. In addition, they show the benefit of smoking cessation among a cohort as a whole, including the continuing smokers with the quitters.

The most straightforward interpretation of ex-smoker data indicating that CHD mortality rates of persons who stopped smoking are substantially lower than those of persons who continued smoking, is that smoking cessation directly results in the reduction of risk of heart disease mortality. Underlying this presumed CHD benefit is the assumption that ex-smokers are a representative sample of smokers, except that they have stopped smoking. If the assumption of representativeness is not valid and significant baseline differences in relevant factors exist between ex-smokers and smokers, then the mortality comparison of ex-smokers and continuing smokers may not properly describe the benefits of smoking cessation for the typical smoker. In the Kaiser-Permanente study (*17*), there were small differences in risk profiles and other factors between those who continued to smoke and those who quit, but these differences were not large enough to account for the differences in CHD death rates.

In summary, each of the several major prospective studies of smoking cessation demonstrates that ex-cigarette smokers have a decreased risk of subsequent mortality relative to continuing smokers. The decreased risk occurs fairly quickly after cessation of smoking, suggesting that the effects of cigarette smoking are reversible. The quitters were self-selected in these observational studies, however, and may include cigarette smokers at lower risk of disease. However, the steadily decreasing risk over time after quitting suggests that more is going on than the simple selection of a lower risk group. Conversely, some smokers may quit in response to symptoms or diagnosis of smoking-related illness, thus possibly

TABLE 2.—Age-, sex-, and race-adjusted death rates according to smoking category and selected major causes

Category	No. of subjects	No. of person-years	Adjusted death rate per thousand person-years[1]					
			All causes	All causes except injuries and poisoning	All neoplasms	Lung cancer	All circulatory diseases	Coronary heart disease
Persistent smokers	9,394	70,348	9.2 (557)	8.1 (485)	3.2 (191)	0.9 (58)	4.0 (240)	2.6 (168)
Temporary quitters	970	6,666	7.1 (46)	6.7 (43)	2.2 (14)	0.9 (6)	3.8 (24)	2.3 (16)
Persistent quitters	2,856	18,798	5.3 (107)	5.0 (102)	1.9 (39)	0.3 (6)	2.2 (46)	1.4 (31)
Never smokers	12,697	99,290	5.1 (569)	4.8 (540)	1.8 (199)	0.02 (2)	2.4 (275)	1.6 (186)

[1] Figures in parentheses denote number of deaths.

Source: Friedmen et al. (*17*).

TABLE 3.—Relative trends in cigarette smoking and coronary heart disease mortality among male physicians in Britain and California in two natural experiments of smoking cessation, where status of other risk factors is unknown

British male physicians, 1951–71[1]

	Time period			
	1951–55	1956–60	1961–65	1966–71
Percentage of physician current smokers at start of time period	41	33	27	21
Ratio of smokers (physicians/British males)	88	68	60	51
	Standardized mortality ratio (physicians/British males)			
CHD and myocard. degen., attained age				
20–54		107	85	62
55–64		120	103	86
65–74		109	100	91
75–84		88	94	100
All causes, attained age				
20–64		82	76	70
65–84		75	77	78

California male physicians, 1950–79[2]

	Time period					
	1950–54	1955–59	1960–64	1965–69	1970–74	1975–79
Percentage of physician current smokers at start of time period	53	48	39	28	20	14
Ratio of smokers (physicians/U.S. males)	100	83	66	55	44	35
	Standardized mortality ratio (physicians/U.S. males)					
CHD	115	97	86	80	74	69
All causes	89	80	79	78	67	67

[1] Study of 34,440 men aged 20+, followed for 20 years. Doll and Peto (*12*).
[2] Study of 10,310 men aged 25+, followed for 30 years. Enstrom (*14*).

underestimating the benefits of quitting that would be expected in an otherwise healthy population. Other variables that may contribute to mortality may not have been included in the analysis.

Randomized Controlled Trials of CHD Prevention Not Involving Smoking Cessation

The most rigorous way to determine the value of smoking cessation is the randomized controlled trial. A series of important experimental or clinical trials have been conducted in the United States and other countries over the past 25 years in order to

establish the effectiveness of primary prevention of CHD through modification of risk factors. These randomized controlled trials involve both primary and secondary prevention (*2*). The primary prevention trials select subjects who are free of CHD or stroke at entry to the study. The secondary prevention trials attempt to modify risk factors after a heart attack or stroke in order to reduce the risk of a second heart attack or death (*6, 7, 8, 38, 40*). Secondary prevention trials and nonrandomized trials are not discussed further here.

Most previous primary prevention trials of CHD have been limited to a single risk factor such as serum cholesterol reduction. Many single risk factor intervention trials include a pharmacologic agent that lowers either serum cholesterol or blood pressure and is compared with a placebo. Most of these studies are further limited to higher risk subjects, such as subjects with serum cholesterol levels in the highest 10 to 15 percent of the population, or to relatively small sample sizes. They did not monitor or control for changes in cigarette smoking habits.

The most extensive primary prevention trials involve dietary reduction of cholesterol; they are described in more detail elsewhere (*2, 39*). The major randomized trials are the Los Angeles veterans domiciliary study (*9*), the Helsinki, Finland, mental hospital study (*42, 59*), and a feasibility study of free-living and institutionalized Americans (*45*). Each of these studies involved about 200 to 400 men in the dietary intervention group and a similar number in the control group.

Another set of randomized trials has involved reduction of high blood pressure using antihypertensive medication—the U.S. Veterans Administration cooperative study (*63*), the U.S. hypertension detection and followup program (*30, 31*), the Australian therapeutic trial (*1*), and the Oslo drug trial (*24*). These large studies followed three small studies—Hamilton et al. (*19*), Wolff and Lindeman (*67*), and the Cooperative Randomized Control Trial (CRCT) (*5*). These studies generally show that lowered blood pressure results in some reduction in CHD among the treated groups relative to the control groups.

Intervention Trials of CHD Prevention Involving Smoking Cessation

The observational epidemiological studies strongly suggest that cigarette smoking cessation decreases the risk of heart attack and CHD mortality compared with the risk for continuing smokers (*60, 61, 62*). All of the observational studies, however, have the limitation that the individuals were not experimentally assigned to smoking and nonsmoking status. Experimental studies such as randomized